Masked Hypertension
A Phenomenon of Measurement

Stanley S. Franklin, Eoin O'Brien, Lutgarde Thijs, Kei Asayama, Jan A. Staessen

Conventional measurement of blood pressure (BP) in individual patient care has several limitations that include causing spurious elevation of BP (white-coat hypertension) or giving normal readings when, in fact, the patient is hypertensive (masked hypertension).[1] Out-of-office BP measurements using home BP monitoring (HBPM) or ambulatory BP monitoring (ABPM) provide insight into the true behavior of BP in individual patients during various daily activities at home and at work and during sleep (with ABPM). Although it is acknowledged at the outset that masked phenomena are a consequence of the methodology of BP measurement, the condition, nevertheless, has serious consequences for the diagnosis and management of patients with hypertension. Recent advances in our understanding of masked hypertension will be examined in this review.

Definitions

Conventional BP in the office or clinic is considered to be normal if it is <140/90 mm Hg. In contrast, out-of office BP values must take into consideration the period of the day simply because BP levels are different during the day and night, and BP may be elevated during either of these periods or throughout the 24-hour period. Indeed the 24-hour period can be further subdivided into the white-coat window (generally the first hour and possibly also the last hour) when the patient is subject to the influence of the medical environment, and the preawakening period when the subject may exhibit a morning surge in BP.[1,2]

Current consensus guidelines define out-of office hypertension as daytime BP ≥135/85 mm Hg, nighttime BP ≥120/70 mm Hg, and 24-hour average BP ≥130/80 mm Hg (Table 1).[1]

The daytime cutoff BP values for hypertension pertain to both ABPM and HBPM. Thus, for daytime measurements, the definition of masked hypertension in untreated individuals is an in-office BP of <140/90 mm Hg and an out of-office BP of ≥135/85 mm Hg. However, as shown in Table 1, outcome-driven ABPM and HBPM cutoff values are generally lower than consensus numbers, mainly reflecting the importance of cardiovascular risk in masked hypertensives that are captured—predominantly in the prehypertension category and occasionally in those with optimal BP.[1–5] Indeed, risk stratification by ABPM across Joint National Classification BP categories, using the International Database on Ambulatory Blood Pressure in Relation to Cardiovascular Outcomes (IDACO) meta-analysis population adjusted for covariables (Figure 1), unmasked masked hypertension in 7.5% and 29.3% of individuals with normotension and prehypertension, respectively.[6,7] The term masked hypertension is reserved for antihypertensive naïve patients. In patients with treated hypertension, the presence of residual masked hypertension is called masked uncontrolled hypertension (MUCH).

Conditions in Which Masked Hypertension Is Likely

Elderly patients with a male predominance who present with increased BP variability are more likely to have masked hypertension than sustained normotension or white-coat hypertension.[8] Conventional office measurement of BP in an elderly hypertensive patient soon after a large meal may produce postprandial reduction of BP and a diagnosis of masked hypertension.[9] Mental stress at work or at home may raise BP to hypertensive levels except at the time of conventional office measurements.[10,11] Smokers and patients who consume excessive alcohol are prone to masked hypertension.[12,13] Sedentary, obese individuals may have poor exercise tolerance throughout the day's activities, whereas they record prehypertensive BP values in the physician's office when measured at rest.[14,15] Finally, the presence of metabolic syndrome, diabetes mellitus, chronic kidney disease, shortened sleep time, or obstructive sleep apnea may predispose to masked hypertension largely because of nocturnal hypertension.[16–20] Indeed, elevated nighttime BP and nondipping or rising nocturnal BP patterns, with or without elevated daytime ABPM values, may be associated with normal conventional office BP values, and hence with a diagnosis of masked hypertension.[20–22] In summary, there is a need for 24-hour ABPM with assessment of both day and nighttime BP to diagnose masked hypertension in the many conditions where it might occur.

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Table 1. Ambulatory and Home Blood Pressure Values (mm Hg)

Category	Interval	Optimal, mm Hg	Normal, mm Hg	Elevated, mm Hg
ABPM				
Consensus	Daytime	...	...	≥135/85
Population	Daytime	<120/80	<130/85	≥140/85
Home				
Consensus	Daytime	...	...	≥135/85
Population	Daytime	<120/75	<125/80	≥130/85
ABPM				
Consensus	Nighttime	...	...	≥120/70
Population	Nighttime	<100/65	<110/70	≥120/70
ABPM				
Consensus	24-h	...	...	≥130/80
Population	24-h	<115/75	<125/75	≥130/80

Consensus and population denote consensus-based and population-based threshold, respectively. ABPM (consensus-based)[1]; ABPM (population-based)[3]; home (consensus-based)[4]; and home (population-based)[5]. ABPM indicates ambulatory blood pressure monitoring.

Progression of Masked Hypertension

There is moderately good prediction and persistence of masked hypertension when patients with prehypertension are screened by conventional office measurements.[23] Pickering et al,[24] early after first describing the entity, thought that masked hypertension, alternating with prehypertension, was a precursor to sustained hypertension. However, a 5-year Quebec population study assessed the persistence of masked hypertension and its progression to sustained hypertension.[25] Among patients with baseline masked hypertension, more than half had either masked or sustained hypertension at 5 years, many of whom were started on antihypertensive treatment; one third progressed to sustained hypertension, one third regressed to normotension, and 1 in 5 remained masked >5 years when not treated. Indeed, delay in making the diagnosis of masked hypertension may account for the high prevalence of hypertensive cardiovascular target organ damage.[21,22,26,27] Furthermore, treated patients with MUCH tend to have persistent target organ damage that is comparable with what is observed in patients with sustained hypertension.[27]

Prevalence of Masked Hypertension

The frequency of masked hypertension varies considerably depending on population characteristics and the presence or absence of antihypertensive treatment. In the 11-country IDACO report of middle-aged and elderly patients (mean age, 64 years), using daytime ABPM, 1168 untreated patients had isolated systolic hypertension: of this total, 314 (26.9%) had sustained hypertension, 334 (28.5%) had white-coat hypertension, and 520 (44.5%) had masked hypertension[28]; importantly, masked isolated systolic hypertensives represented 9.0% of the conventional normotensive group.[28] In another 11-country IDACO report, including 9691 subjects drawn from the general population, nondiabetic patients who were normotensive on conventional measurement had a masked hypertension prevalence of 18.8%, when untreated and 30.5% with treatment (Table 2).[17] In contrast, conventionally normotensive diabetic patients had a prevalence of masked hypertension of 29.3% when untreated and 42.5% with treatment.[17] Thus, the prevalence of masked hypertension not only rises in the presence of diabetes mellitus and in many other high-risk disease states but also is influenced by antihypertensive treatment.

Increased Prevalence of MUCH

In most published reports, the prevalence of masked hypertension is higher in antihypertensive treated when compared with untreated patients; we noted these findings in a recent IDACO study[17] (Table 2); the reasons need to be examined. Antihypertensive treatment on average will lower ABPM values by 60% to 70% of the reduction of conventional in-office BP, ie, approximately a 3 mm Hg systolic BP (SBP) reduction of conventional in-office BP for a 2 mm Hg SBP reduction in out-of-office ABPM.[29,30] More recently, Schmieder et al[31] noted that patients with higher pretreatment SBP levels had an even greater disproportional reduction in office SBP than in ambulatory SBP after undergoing antihypertensive treatment; in addition, these investigators[31] and others[32,33] noted that the white-coat effect decreased by ≈10/5 mm Hg on average after

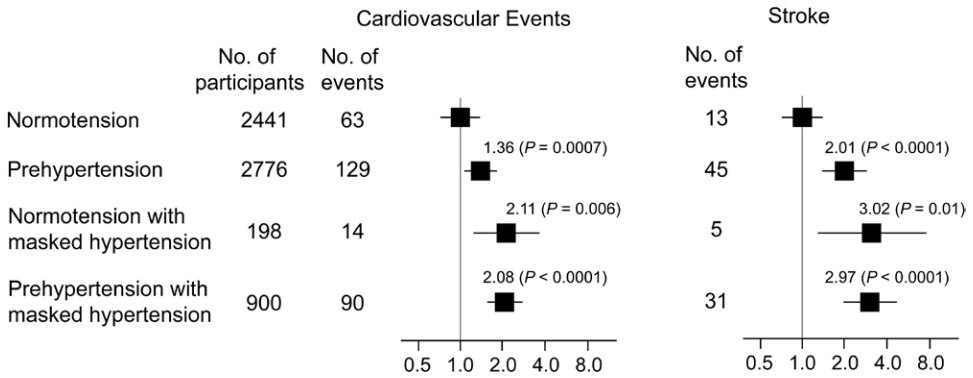

Figure 1. Hazard ratios (HRs) for cardiovascular events and strokes associated with masked hypertension in subjects with normotension (<120/<80 mm Hg) or prehypertension (120–139/90–89 mm Hg) according to their conventional blood pressure.[6] Participants with sustained normotension are the reference group. HRs are obtained from the IDACO (International Database on Ambulatory Blood Pressure in Relation to Cardiovascular Outcomes) database and are adjusted for cohort, sex, age, body mass index, smoking and drinking, cholesterol, diabetes mellitus, and history of cardiovascular complications. Reprinted from Brguljan-Hitij et al[6] with permission of the publisher. Copyright © 2014, the Oxford University Press.

Table 2. Prevalence of Masked Hypertension by Treatment Status in Diabetics and Nondiabetics

	Prevalence, %		Odds Ratio		
Treatment Status	Nondiabetics	Diabetics	Unadjusted	Partly Adjusted	Fully Adjusted
Untreated	18.8% (1031/5486)	29.3% (67/229)	1.79 (1.33–2.40); *P*<0.001	1.46 (1.08–1.98); *P*=0.014	1.35 (0.98–1.86); *P*=0.065
Treated	30.5% (192/630)	42.5% (37/87)	1.69 (1.07–2.67); *P*=0.025	1.59 (1.00–2.52); *P*=0.051	1.59 (0.98–2.58); *P*=0.058

Partly adjusted odds ratios are adjusted for sex and age only. Fully adjusted odds ratios are adjusted for sex, age, conventional systolic blood pressure, history of cardiovascular complications, current smoking status, current alcohol intake, body mass intake and total cholesterol. Reprinted from Franklin et al.[17]

beginning antihypertensive treatment, but was still present after 1 year, thus contributing to the reduction of in-office BP but not in ABPM. Perhaps of equal importance, a morning recording of normal in-office BP may be at peak levels of medication while trough levels later in the day and night may be associated with hypertensive BP values. Regardless of the mechanisms for the disproportional greater reduction in office than in ambulatory BP, the important message for physicians is that treatment aimed at normalizing conventional office BP will increase the percentage of patients with MUCH.

Increased Cardiovascular Risk of MUCH

Not only does antihypertensive treatment increase the prevalence of MUCH but also treatment increases its cardiovascular risk. The effects of antihypertensive treatment versus no treatment on the cardiovascular risk of sustained hypertension, masked hypertension, and sustained normotension in a nondiabetic population are illustrated in a recent IDACO study (Figure 2).[17] There was increased cardiovascular risk in both the treated masked uncontrolled hypertensives and the treated normotensive patients in comparison with the untreated masked hypertensives and untreated sustained normotensive groups, respectively.[17] The logical explanation for these findings is that some patients with sustained hypertension were converted into masked hypertension and some of the masked hypertension patients were converted into sustained normotension. These findings illustrate the epidemiological principle that normalization of BP with treatment does not eliminate lifetime burden associated with previous elevated BP (risk, in part, being dependent on the duration of hypertension), or does it correct the other metabolic risk factors that cluster with the hypertensive state.

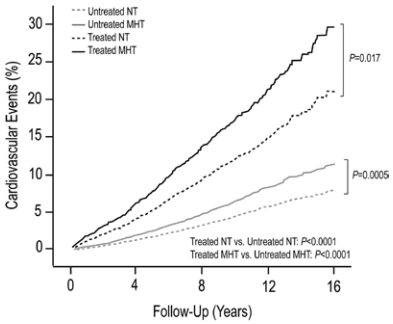

Figure 2. Cohort, sex, and age-standardized incidence of cardiovascular events in untreated and treated normotensive (NT) and masked hypertensive (MHT) nondiabetic subjects that are derived from an IDACO (International Database on Ambulatory Blood Pressure in Relation to Cardiovascular Outcomes) meta-analysis.[17] Fully adjusted hazard ratios (HRs) for treated vs untreated masked hypertensives are as follows: HR, 2.27 (95% confidence interval, 1.6–3.2; *P*<0.0001). Adapted from Franklin et al.[17]

The high prevalence of MUCH among treated subjects suggests that most physicians use suboptimal antihypertensive treatment, largely because they focus primarily on normalizing conventional office BP values. In summary, the conventional normalization of office BP with antihypertensive treatment will leave a significant number of patients with undetected MUCH. In contrast, optimal treatment that uses out-of-office BP monitoring will decrease MUCH by maximal conversion to sustained normotension.

Diagnostic Strategies for Masked Hypertension

Although automated office monitoring of BP is superior to conventional office BP monitoring, it cannot provide a profile of BP over 24 hours as with ABPM or track BP for multiple days, weeks, and months as with HBPM.[34–37] Importantly, a meta-analysis done by Hodgkinson et al[38] concluded that neither office nor HBPM had sufficient sensitivity nor specificity to replace ABPM as the reference standard. A further advantage of ABPM over HBPM is the ability to identify patients with normal daytime but nocturnal masked hypertension. In addition, many of ABPM readings are taken during normal daily activity, whereas HBPM readings are always taken at rest. Consequently, it has been recommended that a positive diagnosis of masked hypertension be confirmed by ABPM before commencing antihypertensive therapy.[1,39,40].

Treatment Strategies for Sustained Hypertension

Del la Sierra et al[41] followed up 2115 treated hypertensive patients from the Spanish ABPM registry for a 4-year duration for cardiovascular events. After adjustment for baseline cardiovascular risk and office BP, nighttime but not daytime SBP predicted cardiovascular events (hazard ratio per SD increase, 1.45; 95% confidence interval, 1.29–1.59). Thus, nighttime BP was the single most important predictor of cardiovascular risk.[41] Using an updated database from The Spanish Society of Hypertension ABPM Registry, Banegas et al[42] identified 31% of patients who had MUCH by 24-hour ABPM (MUCH) among the 14 840 subjects with treated and controlled conventional BP. These patients were well controlled to prehypertensive BP levels with conventional in-office BP monitoring. The clinical characteristic of MUCH patients were male sex, advanced age, obesity, smoking history, diabetes mellitus, and longer duration of hypertension—characteristics that increased the risk of future cardiovascular disease.[42] Importantly, poorer control of nocturnal BP was twice as frequent as daytime ABPM control in defining MUCH and was

the exclusive abnormality in 24% of patients. Indeed, many patients with resistant MUCH had persistent failure to control nighttime hypertension.

Furthermore, Banegas et al[43] suggest that the lack of benefit of antihypertensive treatment in some randomized control trials may partly result from excluding some patients with masked hypertension and including some patients with white-coat hypertension at trial baseline. Consequently, the use of ABPM in clinical trials may lead to a better assessment of trial eligibility.[43]

Treatment Strategies for Masked Hypertension

There is strong evidence that masked hypertension patients have increased risk of target organ damage, cardiovascular, and renal morbidity[1,27,44] with an overall cardiovascular risk approaching that of sustained hypertensives[27]; therefore, one can make a good case for starting antihypertensive therapy when the diagnosis of masked hypertension has been substantiated. What is lacking, however, is a randomized controlled trial that assesses the optimal level of daytime and nighttime BP reduction to weigh therapeutic benefits versus cardiovascular risks. There are additional questions that remain unanswered: 1) Because in-office BPs are already well within normal guidelines in patients with masked hypertension, is there a limit to further reduction in-office BP that would pose a cardiovascular risk? (2) Will the benefit of active drug treatment in reducing hypertensive target organ damage and cardiovascular events be similar in patients with masked hypertension as in those with sustained hypertension?

Perspective

Whereas masked hypertension is a consequence of the methodology of BP measurement, the condition, nevertheless, has serious consequences for the diagnosis and management of patients with hypertension. Not only does masked hypertension occur in a multitude of diverse clinical settings that on frequent occasions elevate BP more often at night than during the day but also these associated medical conditions are at high risk for future cardiovascular disease events. Importantly, initiating antihypertensive treatment with only conventional office BP assessment may transition sustained hypertension into a high percentage of MUCH, rather than the desired therapeutic goal of sustained normotension. Quantitatively, the exclusive use of in-office BP to determine how low to go will undertreat ≈1 out of 3 patients who will be left with MUCH. Importantly, 2 out of 3 of these insufficiently treated patients will have nocturnally driven masked hypertension that requires ABPM for detection. Therefore, although HBPM may be the preferred diagnostic method of assessing out-of-office BP during the initiation and dose-titration of antihypertensive therapy, the use of ABPM will be necessary to rule out undiagnosed nocturnal masked hypertension. Importantly, undiagnosed and untreated masked hypertension and treated but uncontrolled masked hypertension represent 2 significant high-risk populations of public health concern. Indeed, current evidence strongly supports revision of National and International guidelines that base diagnostic and therapeutic decisions on patient's age and conventional in-office levels; these measurements should be supplemented with HBPM and ABPM to address the current worldwide poor control-rate of hypertension.

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Disclosures

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